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PCOD – AYURVEDIC REVIEW AND MANAGEMENT

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ABSTRACT

Woman as beautiful as nature, is the most delicate, complicate and supreme creation of god to mankind. The transition of a girl to a woman and a woman to a mother adds a sense of fullness to her life. Woman unlike man is subjected to various diseases in relation to her reproductive system. One such disease is POLYCYSTIC OVARIAN DISEASE (PCOD). PCOD is a disease which is related to the cystic changes in the ovary. The follicles develop from primordial follicles but the development stops at an early antral stage due to the disturbed ovarian function. PCOD manifests clinically in many ways. 1) Amenorrhoea /Oligomenorrhoea 2) Hirsutism 3) Alopecia 4) Acanthosis nigricans 5) Acne 6) Anovulation. Anovulation is the major pathology that is responsible for various changes in PCOD. PCOD in Ayurveda can be considered as *rasa medo dhatu vikara* and has to be managed depending on the *dosha dushya* vitiation. Ayurvedic management principles as *amahara chikitsa*, *sodhana* and *samana* therapies along with *vata kapha hara dravyas*, diet and lifestyle modification proves to be beneficial in the management of PCOD. A few drugs possessing the above properties have been described here and their clinical application had helped in the management of PCOD.

Keywords: PCOD, *rasa medo dhatu vikara*, Hirsutism, Anovulation, *Samana*.

INTRODUCTION

Woman as beautiful as nature, is the most delicate, complicate and supreme creativity of God to mankind. Her enormous physical and psychological energy excels her in both family as well as professional life. Her unique capacity of attaining menarche, maintaining a fertile age up to menopause is the testimony of womanhood. The transition of a girl to a woman and a woman to a mother adds a sense of fullness to her life.

A woman should be very cautious as it is high time that may be the delicacy of that precious womanhood is in trouble. Woman unlike man is subjected to various diseases in relation to her reproductive system. These diseases may be simple that can be

managed easily with simple medication whereas a few of them may need a long term management. They may also land up a woman in various complications. One such disease is POLYCYSTIC OVARIAN DISEASE (PCOD).

The condition was first described in 1935 by the American gynecologists Irving F Stein and Michael L Leventhal, from whom its original name of Stein-Leventhal syndrome was taken. The earliest published description of a patient with what is now recognized as PCOS was in 1721 in Italy. Cyst-related changes to the ovaries were described in 1844. [1]

The syndrome acquired its name due to the common sign on ultrasound examination of multiple ovarian cysts which represent immature follicles. The follicles have developed from primordial follicles but the development has stopped at an early antral stage due to the disturbed ovarian function. The follicles may be oriented along the ovarian periphery appearing as a 'string of pearls' on ultrasound examination.[2]

Androgen excess PCOS Society criteria: In 2006, the Androgen Excess PCOS Society suggested a tightening of the diagnostic criteria to all of the following including excess androgen activity, oligoovulation/ anovulation, polycystic ovaries and other entities are excluded that would cause excess androgen activity.[3]

Aim and Objectives:

1. To understand the pathology of polycystic ovarian disease in Ayurveda
2. To evolve the management principles.
3. To enlist certain Ayurvedic drugs that would be beneficial in the management of the polycystic disease.

Materials & Methods:

Various Samhitas, modern medical text books and websites.

DISCUSSION

PCOS produces symptoms in approximately 5% to 10% women of reproductive age (12–45 years old). It is thought to be one of the leading causes of female sub fertility and the most frequent endocrine problem in women of reproductive age.[4]

The predisposing factors for PCOS include the following.[5]

- Genetic factors
- Family history of PCOS
- High maternal androgen: Prenatal exposure to androgens poorly controlled maternal congenital adrenal hyperplasia, Androgen-secreting tumors, and low birth weight/small for gestational age, premature adrenarche.
- Endocrinal factors: Onset of type 1 diabetes mellitus before menarche, insulin resistance and obesity.
- Drugs: such as anti-epileptic drugs (e.g., Valproate)

Clinical features of polycystic ovary syndrome[6].

1. Oligomenorrhea/amenorrhea
2. Infertility/first trimester miscarriage
3. Obesity
4. Hirsutism
5. Acne
6. Acanthosis nigricans
7. Male pattern alopecia

Anovulation is the major pathology that is responsible for various changes in PCOD.

Pathology:

Patients with PCOS have higher gonadotrophin releasing hormone (GnRH), which in turn results in an increase in LH/FSH ratio in females with PCOS. The majority of patients with PCOS have insulin resistance and/or obesity. Their elevated insulin levels contribute to or cause the abnormalities seen in the hypothalamic-pituitary-ovarian axis that lead to PCOS. Hyperinsulinemia increases GnRH pulse frequency, LH over FSH dominance, increased ovarian androgen production, decreased follicular maturation and decreased SHBG binding. All these factors contribute to the development of PCOS [7, 8].

PHYSIOLOGY	PATHOLOGY
Hypothalamus ↓ GnRH ↓ Ant. Pituitary (FSH. LH) ↓ Decrease in receptor sensitivity of GnRH	Hypothalamus ↓ GnRH ↓ Ant. Pituitary (FSH. LH) ↓ Increase in receptor sensitivity of GnRH

PHYSIOLOGY	PATHOLOGY
FSH + LH ↓ Growth & maturity of follicle ↓ Maturation of Oocyte **LH** ↓ Stimulates Theca cells Androgens ↓ Estrogens ⇄ **LH surge** **Suppresses FSH** (Negative feedback)	⊙ **FSH + LH** 1 : 3 FSH ↓ Maturity of follicle disturbed **LH** ↓ Stimulates Theca cells Androgens **Defective Aromatisation** ↓ Estrogens (Positive feedback)

PHYSIOLOGY	PATHOLOGY
ESTROGENS+LH surge ↓ **OVULATION** ↓ Ruptured follicle ↓ CORPUS LUTEUM ↓ ↓ Progesterone Estrogens **Estrogen** → 20% Albumin, 69% SHBG, 1% Biologically active Max. bounded Estrogens	Tonically elevated LH + Min FSH ↓ **ANOVULATION** ↓ **Min** Progesterone ↓ Increased ANDROGENS ↓ Decreased **SHBG** Max. Unbounded Estrogens

PHYSIOLOGY	PATHOLOGY
Progesterone + Estrogen Max. Upto 1 wk after ovulation (Suppresses FSH +LH)	Unbound Estrogens ↓ Hyper Estrogenic State ↓

.Wanes after 1 wk ↙ ↘ Menses Stimulates Hypothalamus ↘ GnRH release	Positive feedback on Anterior Pituitary ↓ Increased sensitivity to GnRH

It should always be borne in mind that a single cyst is never a diagnostic feature of PCOS. Ovary should contain multiple cysts with increased ovarian stroma. Moreover in PCOS the periods do not stop abruptly but takes a gradual course of time.

INVESTIGATIONS:

Laboratory tests

1. Serum levels of androgens, including androstenedione and testosterone may be elevated. Dehydroepiandrosterone sulfate (DHEA-S) levels above 700-800 mcg/dL are highly suggestive of adrenal dysfunction. The free testosterone level is thought to be the best measure, with about 60% of PCOS patients demonstrating high levels[9]
2. The ratio of LH (Luteinizing hormone) to FSH (Follicle stimulating hormone) is greater than 1:1 (sometimes more than 3:1), as tested on day 3 of the menstrual cycle [10]
3. Fasting biochemical screen and lipid profile[11].
4. Two-hours oral glucose tolerance test (GTT) in patients with risk factors (Obesity, family history, history of gestational diabetes) may indicate impaired glucose tolerance (insulin resistance) in patients with PCOS [12]
5. Fasting insulin level or GTT with insulin levels (also called IGTT): Elevated insulin levels have been helpful to predict response to medication and may indicate patients who will need higher doses of metformin or the use of a second medication to lower insulin levels. A hypoglycemic response in which the two-hour insulin level is higher and the blood sugar lower than fasting is consistent with insulin resistance. [13]

Ultra-sonography:

It looks for small ovarian follicles. According to the Rotterdam criteria, 12 or more small follicles should be seen in an ovary on ultrasound examination. The numerous follicles contribute to the increased size of the ovaries that is 1.5 to 3 times larger than normal[14].

Differential Diagnosis of PCOS

Other causes of irregular or absent menstruation and hirsutism, such as hypothyroidism, congenital adrenal hyperplasia (21-hydroxylase deficiency), Cushing's syndrome, hyper-prolactinemia, androgen secreting neoplasms and other pituitary or adrenal disorders should be investigated. PCOS has been reported in other insulin-resistant situations such as acromegaly. [15]

PCOD is a syndrome rather than a disease. The complications include Infertility, Obesity and Type-II Diabetes.

AYURVEDIC PERSPECTIVE

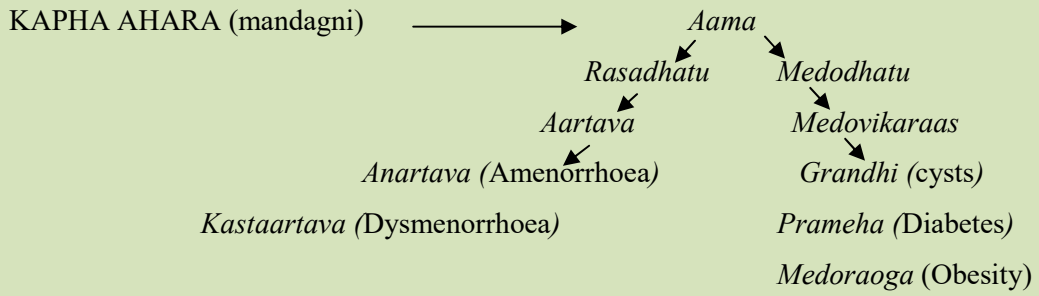
Ayurveda tries to maintain all the humoral functions of the body in a normal state. The functions of *Kayagni*, *Bhutagni* and D*hatvagni* include all the meatabolic, neuronal and hormonal functions of the body. Hence Ayurveda stresses at maintaining proper A*gni* as a prime factor in the management of many diseases.

Due to *Kapha vardhaka ahara* and *Vihara*, *Mandagni* results in. This *Mandagni* results in the formation of *Ama*. *Kapha* along with the *Ama* first vitiates the *Rasa dhatu*. As *Artava* is the *Upadhatu* of *Rasa dhatu*, the *Artava* is vitiated. The vitiated *Kapha* along with the *Ama* causes obstruction to the *Artava* in the *Artava vaha srotas* there by leading to *Anartava* (Amenorrhea) or *Alpa artava* (oligomenorrhea).

Aggravated *Kapha* and *Ama* having vitiated *Rasa dhatu* moves towards *Medodhatu* as *Kapha*, *Ama* and *Medas* have similar qualities. Hence they are attracted to each other. *Medo dhatu* is one of the first *dhatus* along with *Rasa dhatu* to reflect a *Kapha* aggravation.

Medodhatvagni being affected by the presence of increased *Kapha* and *Ama* causes *Medovriddhi* (obesity). This results in the manifestation of *Medo vikaras* as *Granthi* (cysts), *Prameha* (diabetes) and *Medo roga* (obesity).

PCOD AN AYURVEDUC APPROACH



Samprapti Ghatakas

- *Doshas - Kapha , Vata & Pitta*
- *Dushyaas - Rasa-Medo-Artava*
- *Srotas - Rasa-Medo-Artava*
- *Sroto Dusti - Granthi & Sangam*
- *Vyadhi Adhistanam – Garbhasaya*

Ayurvedic Management:

- *Aama Chikitsa*
- *Vatakapha Hara Dravyas*
- *Sodhana*
- *Samana*

***Aama Chikitsa*:** [16]

"Pachanair deepanai snehaistaan swedaischa parishkratan
Sodhayeth sodhanaih kale yadhaasannam yadha balam"

Deepana and *pachana* drugs as *chitrakadi vati* has to be given. *Snehana, swedana* and then *sodhana* according to the *bala* of the patient has to be done.

VATAKAPHA HARA DRAVYAS

1. ***Apamarga****: Deepana, Pachana and kapha medo anilaapaha*[17]
2. ***Karanja****: Deepana, Pachana, Vatakaphahara and especially Yonidosha hrit*[18]
3. ***Jeeraka & Krishna jeeraka****: Deepana, Pachana, Vatakaphahara and Garbhasaya visuddha krit*[19]
4. ***Guggulu****: Deepana, Amahara, Kaphavatahara, Medohara, Pramehahara, Granthi hara*[20]

Sodhana Chikitsa

Uttara vasti with *Dhanvantara taila* will be beneficial.

Samana Chikitsa

1. ***Kalyana ghritam****: swalpa rajasi (oligomenorrhea), Pramehahara and Pumsavaneshucha* [21]
2. ***Pushyanuga churnam****: Yoni dosha hara, Rajo dosha hara* [22]

CONCLUSION

Hence PCOD in Ayurveda can be considered as *rasa medo dhatu vikara* and has to be managed depending on the *dosha dushya* vitiation. Ayurveda is blessed with many drugs which help to combat this disease in a more natural way. *Deepana* and *pachana* drugs as *chitrakadi vati* help to alleviate *ama*. This increases the digestive capacity of the patient which is the first step in the management of PCOD. *Vata Kaphahara* and *Medohara dravyas* property helps to regulate the *apanavata* and check *medodatu vikrutis* like *grandhi, prameha* and *sthoulya*. *Kalyana ghrita* and *Pushyanuga churnam* are useful in oligomenorrhoea.

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